

# Exercise training in chronic heart failure: why, when and how

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## Summary

The management of patients with chronic congestive heart failure has changed considerably during the last decade. Until recently, restriction of physical activity was recommended for patients with chronic heart failure. However, the knowledge that training influences largely the periphery rather than the heart itself has led to a dramatic change in the approach toward training in patients with chronic heart failure.

*Why to train patients with chronic heart failure:* Training increases exercise tolerance by an average of 20% in chronic heart failure regardless of etiology (ischemic or non-ischemic cardiomyopathy) or severity of left ventricular dysfunction. Available data, while limited, demonstrate that increases in exercise capacity are paralleled by an improvement in quality of life.

Studies have consistently demonstrated that training has no deleterious effect on central haemodynamics, left ventricular remodeling, systolic or diastolic function, or myocardial metabo-

lism. At present, there are insufficient data to determine the effect of training on prognosis, but trials are currently underway to address this.

*When to train patients with chronic heart failure:* Exercise training should be performed only with the patients that have been in a stable clinical condition for a period of at least 3–4 weeks. Clinical stability is defined as no change in symptoms, weight, drug regimen, or NYHA class over this period.

*How to train patients with chronic heart failure:* Initially, the program should be supervised for a period of 2 to 4 weeks; home-based programs are usually appropriate thereafter. Activities that can be maintained for a lifetime should be encouraged, and the focus should be on aerobic-type activities. The intensity level should be targeted to about 50%–70% of peak  $\text{VO}_2$  and/or Borg ratings of 12–14 (“walk and talk”).

*Key words:* chronic congestive heart failure; exercise training

## Introduction

During the last decade, the management of patients with chronic congestive heart failure has changed dramatically. In the 1988 edition of Braunwald's textbook “Heart Disease”, restriction of physical activity was recommended for patients with chronic heart failure [1]. In recent years however, knowledge about the pathophysiology of heart failure has increased considerably. For instance, it has been demonstrated that exercise tolerance in patients with chronic heart failure is poorly correlated with measures of left ventricular performance. In fact, it is now appreciated that

there is a better relationship between exercise tolerance and peripheral abnormalities occurring within the framework of chronic heart failure [2–5]. The knowledge that training influences mainly the peripheral muscle and not the heart itself led to a dramatic change in the approach of physicians and researchers toward training in patients with chronic heart failure. This paper reviews the data published in the literature and discusses the major issues related to “Why, When and How” to train patients with chronic heart failure.

## Why to train patients with chronic congestive heart failure

### Influence of training on exercise capacity and quality of life

Patients with chronic heart failure suffer from decreased exercise capacity, impaired quality of

life, and poor prognosis. Treatment strategies are generally targeted to influence these parameters. Medical therapy can improve quality of life and prognosis, but the results of studies assessing the

effects of medical therapy on exercise capacity have been inconsistent. On the other hand, exercise training has repeatedly been shown to improve exercise capacity significantly [6–10].

Ten major randomized trials assessing the influence of training on exercise capacity with gas exchange measurements have been published. All have reported a significant increase in maximal exercise capacity, and some have also reported significant improvements in submaximal endurance or 6 minute walk performance [11, 12]. The average increase in peak  $\text{VO}_2$  in these studies is about 20%, and it has been demonstrated that this improvement in exercise capacity can be sustained over one year [13]. It is important to note that subgroups of chronic heart failure patients who have undergone exercise training have not improved their peak  $\text{VO}_2$  [14–16], and that these patients are difficult to predict [17]. By comparison, ACE-inhibitors improve exercise capacity by only an average of roughly 10% [9]. Importantly, the increase in exercise capacity appears to be paralleled by symptomatic improvement. Coats et al. reported improvements in measures of fatigue, breathlessness, ability to perform daily activities, and general well-being after exercise training in a controlled study [2].

#### **Influence of exercise on central haemodynamic adaptations, left ventricular remodeling, systolic and diastolic function, and myocardial high-energy phosphate metabolism**

An important point to consider is whether training has any additional deleterious influence on the cardiovascular system. Any cardiovascular intervention has the potential for a multitude of adaptations which include a possible deleterious effect. This question has generated a great deal of controversy over the years. Today's technology enables the analysis of key parameters of the cardiovascular system to assess potential negative influences of training. These key parameters include changes in central haemodynamic responses, LV remodeling, systolic or diastolic function, and myocardial high energy phosphate metabolism. Each of these is discussed in the following.

#### **Influence of training on central haemodynamic adaptations**

Four major trials have studied the influence of exercise training on central haemodynamic adaptations [5, 14, 18, 19]. All but one study used a controlled design. Only studies with invasive measurements are considered here. A total of 74 patients have been included in the four studies. The invasive measurements at rest and at maximal exercise were performed upright in three and in the recumbent position in one study. Pulmonary wedge or pulmonary artery pressures were unchanged after training in three trials, but increased significantly in one. The reason for that increase remains unclear, although it could be due to a difference in body position during the measurements.

This may also explain why cardiac output failed to increase after training in that study, whereas it increased in the other studies. Peripheral resistance was reduced in 3 of the 4 studies.

While clearly more studies are needed in this area, the available evidence does not suggest that training causes any worsening of central haemodynamic responses. Indeed, 3 of the 4 studies have demonstrated that training improves cardiac output responses to exercise, and does not elevate intrapulmonary pressures.

#### **Influence of training on left ventricular remodeling**

Progressive left ventricular dilatation often occurs after a myocardial infarction [20, 21]. The combination of ventricular wall thinning, aneurysm formation, expansion of the infarct area, and an increase in the radius of the left ventricle has been termed "ventricular remodeling" and appears to represent an important prognostic marker [22]. The mechanism for ventricular remodeling is unknown, but several factors appear to be involved [23]. Until the late 1980s, it was argued that training could adversely influence the remodeling process. Indeed, some animal studies have demonstrated further ventricular dilatation with training [24, 25], while others have shown favourable effects on left ventricular remodeling [26]. Jugdutt et al. [27] reported a significant deterioration of both global and regional ventricular function after 12 weeks of exercise training in a program that was initiated 15 weeks after an acute anterior myocardial infarction in humans. However, this study was carried out in a small number of patients and was not randomised. In addition, standardisation and timing of exercise training was lacking, and the topographic indices of the left ventricle were derived only from the echocardiographic short axis view.

A total of 6 subsequent studies in humans, among more than 150 patients with both ischemic and non-ischemic heart failure and both moderate and severe left ventricular dysfunction, failed to confirm these results [19, 28–32]. These subsequent studies clearly demonstrated that training has no adverse effects on the LV remodeling process. In fact, Giannuzzi's second study even suggested that training had a beneficial influence on left ventricular remodeling, in that LV global and regional dilatation appeared to be attenuated after two years of training [31]. The contrasting results obtained by Jugdutt et al. [27] and subsequent studies are difficult to explain, but they may be due to differences in patient population, intensity of training and perhaps most importantly, measurement techniques.

#### **Influence of training on systolic and diastolic function**

In the clinical setting, assessment of LV systolic function has, for the most part, been limited to measurements of LV ejection fraction, and the

available evidence suggests that the improved exercise capacity after training in chronic heart failure is accomplished without any detectable impact on LV systolic properties [33]. However, ejection fraction is neither a sensitive nor a specific parameter to detect subtle changes in ventricular function. Indeed, the contraction and relaxation processes of the left ventricle are complex. In addition to the shortening or radial displacement (ejection fraction), the heart moves towards the apex (translational motion) and shows rotational movements [34]. Thus, for the assessment of the functional status of the left ventricle, rotational and translational changes in addition to ejection fraction should be taken into account. Recently, novel myocardial tagging techniques using MRI have been developed, which make it possible to label specific myocardial regions non-invasively and quantify radial displacement and translational and rotational movements.

We have applied this technique in 25 patients with non-ischemic cardiomyopathy randomised to a training or to a control group. Rotation velocity did not change significantly over the study period, suggesting that systolic function is not influenced by training. However, relaxation velocity increased significantly in the exercise group after training. Recent observations demonstrating a slowed relaxation velocity among patients with outflow obstruction, post-infarction, and hypertrophic cardiomyopathy using similar technology suggest that more rapid relaxation is associated with improved diastolic function. This is in agreement with the data published by Belardinelli et al., who found an improvement in echo-doppler measurements of diastolic properties after training [35].

### **Influence of training on myocardial high energy phosphate metabolism**

Changes in myocardial high energy phosphate metabolism have been shown to be related to the severity of left ventricular dysfunction. We have recently studied the influence of exercise training on myocardial high-energy metabolism, and the PCr/ATP ratio in patients with chronic heart failure. Our preliminary results indicate no significant change in relative values of high-energy phosphates in the myocardium due to training. However, the relation between PCr/ATP and  $\text{VO}_2$  at the lactate threshold improved significantly after training [36]. These results suggest that exercise training not only does not harm myocardial energetics, but indicates that myocardial work is performed more economically after training.

### **Influence of training on prognosis**

At present, there is insufficient evidence to provide a conclusive answer regarding the influence of training in chronic heart failure on prognosis. The limited available data do however suggest a favorable effect on prognosis [17]. Training-induced changes such as improved endothelial function, increased threshold for ventricular fibrillation, and changes in autonomic tone, along with limited data from clinical trials [28], suggest that training may evolve to have an important role in prognosis.

### **Influence of training on other important variables**

Training can improve skeletal muscle blood flow during exercise [14], increase mitochondrial density of the skeletal muscle [5], reduce ventilatory abnormalities [37], reduce the activity of the sympathetic and renin-angiotensin system [15], and influence cardiac cachexia by preventing a decrease in growth hormone [38].

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## **When to train patients with chronic congestive heart failure**

An exercise training program should be started only with the patients who are in a stable clinical condition since at least 3–4 weeks. Clinical stability is defined by stable symptoms (no change in NYHA class) and stable fluid balance. Caution is appropriate when systolic blood pressure is below 80 mm Hg at rest, resting heart rate is below 50 beats/min or above 100 beats/min [39].

A cardiopulmonary exercise test should be performed before starting a training program. Relative and absolute contraindications to exercise training among patients with stable chronic heart failure, as recently outlined by a working group report of the European Society of Cardiology and the AACVPR, should be considered [39, 40] (see table 1).

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## **How to train patients with chronic congestive heart failure**

Recommendations for exercise training in chronic heart failure should consider the particular pathology of the patient, the individual re-

sponse to exercise, and the gas exchange data obtained during cardiopulmonary exercise testing prior to training. Both aerobic and strength train-

**Table 1**

Inclusion and exclusion criteria for training of chronic congestive heart failure patients.

Include	
Stable chronic congestive heart failure	
Minimal peak VO <sub>2</sub> of 10 mL/kg/min	
Optimal medical treatment	
Exclude	
Active viral or autoimmune myocarditis	
Obstructive disease (valvular, subvalvular)	
Serious arrhythmias	

**Table 2**

General guidelines for training patients with chronic congestive heart failure.

Initially (2-4 weeks) institution-based training
Dynamic, aerobic exercise or individualized programs (interval, strength, pulmonary muscle training)
Training intensity: 50 to 70% of peak VO <sub>2</sub> or Borg 12 to 14
Duration: 30-45 min per session
Frequency: 3-5 sessions per week for at least 4 weeks, then home based for life

ing are now recommended, although the latter should focus only on low resistance, high repetition exercises, and are not appropriate in all chronic heart failure patients [40]. Training programs in chronic heart failure patients should initially be supervised by trained personnel (initial 2-4 weeks), and thereafter can generally be home-based [39]. A subgroup of high risk patients may require continued supervision; the AACVPR has outlined patients who require continuous ECG monitoring [40].

Exercise programming must be guided by the individual goals, capacities, and needs of the patient. Clearly, impaired exercise tolerance, and reduced prognosis are the main objectives that can be favorably influenced by training.

### Training modalities for increasing exercise tolerance

Any training session should begin with a warm-up period. A warm-up period raises the metabolic rate gradually, begins distributing blood flow to the muscles and joints, and improves flex-

ibility and range of motion. Studies suggest that aerobic exercise (walking, jogging, cycling) is more effective than strength training in increasing physical capacity [39]. A significant increase in exercise tolerance can be achieved after 4-8 weeks with moderate exercise intensity (50%-70% of initial peak VO<sub>2</sub>) [19], and with either continuous or interval training. Appropriate interval training protocols that have been used for patients with chronic heart failure involve rest periods (e.g. walking) interspersed with periods of relatively higher intensity exercises, typically employing an exercise to rest ratio of approximately 1 to  $\geq 2$ . An important component however, is the training duration. Studies have shown that exercise capacity is significantly reduced after only 2-4 weeks of physical inactivity after successful exercise training [12, 41]. Thus, patients should be encouraged to maintain a physically active lifestyle after the training program.

In summary, training modalities for increasing exercise tolerance should be based mainly on aerobic exercise mixed with some strength training, with the modality (jogging, cycling, swimming etc.) being individualised to each patient. Training intensity should be adapted to the individual exercise capacity of each patient; perceived exertion ratings in the range of 12-14 are generally appropriate. The physical activity program should be designed such that it can be performed life long at a moderate intensity (table 2).

### Training modalities for improving prognosis

No data exist at the present time concerning the prognostic implications of different training modalities, intensities, or duration in chronic heart failure patients. However, evidence suggests that regular physical activity can favorably influence prognosis [41].

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